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HYPNOSIS IN MEDICAL PRACTICE

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Hypnosis has been defined as an "artificially induced state, usually, although not always, resembling sleep, but physiologically distinct from it, which is characterized by heightened suggestibility as a result of which certain sensory, motor and memory abnormalities may be induced more readily than in the normal state." To James Braid, an English surgeon, we are indebted for the coining of the word.

The production of a state of hypnosis as a therapeutic measure is a form of direct active intervention in a psychotherapeutic process which in many ways is the antithesis of the non-intervening, listening approach. In some instances, however, such non-intervention may bring about a reaction in the patient of a strong, positive transference which may simulate very closely that of the hypnotized patient.

The **historical background of hypnosis** is significant for an understanding, not only of the process, but of psychotherapy itself. The ancient peoples, Egyptians, Greeks, Romans and Asiatics, were familiar with the hypnotic state. Hypnotic suggestive influences for the cure of disease were also used in medieval times. Mesmer's development of hypnotic techniques, towards the end of the 18th century, represent its first significant development as a form of therapy in modern times. Charcot, Bramwell, Bernheim, Liebauet, Forel and Janet are a few of many modern physicians who used hypnosis as a psychotherapeutic tool and technique of investigation.

Freud's psychoanalytic technique as well as some other psychotherapeutic methods have roots in these hypnotic therapies of the nineteenth century, carried out so extensively in the management of functional disorders. The relatively recent interest in hypnosis in the U.S.A. in hypnoanalysis, hypnocatharsis, and in obstetrics and surgery constitutes a revival of nineteenth century European medical practices with considerable advance in an understanding of the significance of the hypnotic state.

James Esdaile, an English surgeon practicing in India during the middle years of the 19th century, regularly employed hypnotic techniques even in major operations to inhibit pain. This usage in medical and dental surgery, as well as obstetrics, continues to the present day, though not accepted in medical practice as a standard procedure. The phenomenon of post-hypnotic suggestion, as an induced involuntary control over another person's behavior, was extensively studied by Bernheim who called it "latent memory."

Details of technique for inducing hypnosis may be found readily in many books on hypnosis. Acquisition of the

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technical skills for inducing a trance are easily acquired. Every psychotherapist should be familiar with some of the techniques used to induce the necessary state of relaxation. Then, there follows induction of a state of response to external influences from which the patient progresses to a degree of suggestibility in which a sleep-like state, hallucinations and delusions, motor phenomena of increased contraction or paralysis of the muscles, a catatonic-like state, and altered states of sensibility can be produced. The criteria for deep hypnosis are loss of sensation, paralysis, hallucinations, false beliefs, and the carrying out of post-hypnotic suggestions in response to the influence of a therapist.

Experimental Work

Hypnosis has also experienced some usage in scientific and clinical experimental work. Richet, Heindenheim, Preyer, Mobius, Bleuler, Moll, Brodman, and Vogt, operating in neurological and psychiatric clinical laboratories of a number of European universities, employed responsiveness to hypnotic influences as a crucial factor in the differential diagnosis between neurological and functional illness. They also distinguished among levels of hypnotic induction. By and large, they agree on three stages, though a variety of terms are used for each.

About 35% of their population were completely unresponsive. About 85% of their subjects could reach only the first level: somnolence, dreamy state, slight, or lethargy stage. A middle stage (hypotoxic, catatonic, deep or catelepay) was attainable by approximately 45% of their subjects, and the third stage of somnambulism, or hypnotic coma, was reached by only 20%. Children between the ages of 8 and 12 are the best subjects, the extroverted are more responsive than the introverted, and female better than male.

The **nature of hypnotic phenomena** needs discussion in some detail. In order to have a suitable theory for the hypnotic experience, one must, as White indicated, explain a number of phenomena such as (1) the ability to transcend voluntary capacity, as in insensitivity to pain; (2) the peculiar manner in which action is carried out differently from the waking state, in that behavior is humorless and without surprise or apology for bizarrity, as if the individual were insulated from the environment in an abstract, inattentive, unselfconscious way; and (3) why the simple procedure which produces hypnosis brings about such an unusual state. Modern psychological concepts of goal directed behavior explain the process of hypnosis in keeping with neurophysiological, psychoanalytic, and psychologic concepts of behavior.

It is very difficult to understand hypnosis on a neurophysiological basis. The cortical inhibition which can be produced in animals, as shown by Pavlov, has been likened to the hypnotic state. He looked upon this behavior as a response to an overwhelming superior force to be thought of as a means of saving life, since an immovable object is less noticeable and less likely to provoke an aggressive reaction in an annihilating agent.

Concept of West

In a neurophysiologic explanation given by West, the emphasis is placed on the dissociation of awareness from the majority of sensory events taking place. This phenomenon characterizes many dissociative states such as dreams, hypnogogic states, sleep paralysis, highway hypnosis, trances, ecstasies in religious rites, fixation in flyers and day dreaming as well as hypnosis. The diminished input of sensory information in the presence of continued alertness permits an increasing awareness of the brain's previously recorded perceptions. The brain system for the variable integration of incoming stimuli and states of awareness is the combination of ascending reticular activating system, the thalamic reticular formation and the thalamocortical projection system. This network is involved in integrating sensory information, arousal, emotions and memories.

West goes on to theorize that "When there occurs a marked narrowing of focus of awareness on a particular aspect of reality, to the exclusion of much of the rest (as in initiation of the hypnotic trance), then sensory data (verbal suggestions) taken in through an open channel (rapport) become effective because

they are not subjected to ordinary reality testing." A similar phenomenon occurs when arousal is high (strong emotion), during which a functional non-acceptance of additional incoming information takes place.

Influence of Experience

The psychological substrata which form the basis for hypnosis comes from the past experiences during infancy and childhood which have led the individual to believe (1) that he is helpless and dependent on external influences for control and guidance; (2) that he will not be helped, loved or wanted unless he behaves, submits, is obedient and follows directions; (3) that to avoid punishment, injury or rejection, it is necessary to carry out the orders of others; and (4) that future rewards can be expected for the negation of delaying of immediate satisfactions. The patient strives to achieve the goal of pleasing the hypnotist to gain his love and affection and avoid his hostility and rejection for one or many reasons inherent in the total situation within which the hypnosis is being carried out. The hypnotic experience is a regressive phenomenon in that the behavior is a return to earlier patterns of reacting to authority. It is characterized by compliance.

The early infantile and childhood patterns of responding represent the patterns which had efficient adaptive values at the time. They represent what seemed to be the best solution. For instance, being a "good boy" may have been the best possible solution to an overwhelming rigid and punitive atmosphere. This permitted maximum gratification of wishes and desires with a minimum threat of danger in terms of loss of love or security (receiving physical care, i.e., food, shelter, and reasonable protection against physical violence). When behavior is found to be adaptively valuable, the responses become stereotyped, repetitive and inflexible, and do not require conscious awareness of the total situation in which they occur. Shortening of the distance between the stimulus situation and response occurs. This is illustrated by the child who might respond to mother's request to control bowel functions out of fear of loss of love. This same child later develops patterns of obedience to the mother with less conscious awareness of a fear of losing his mother's affection and he relates his obedience to a desire to be good and to the fact that he personally wants to and feels it desirable to control bowel function. In new situations responses are made in keeping with principles of economy—the previously effective and most used patterns are repeated. Because of the long period of dependency in the development of the individual, the tendency toward regression, passivity, dependence and wish-fulfilling phantasy are strongly ingrained.

As indicated, the early infantile magical expectations from parents and people in authority remain as an experiential background which can be revived in the present. Experiences which tend to create a susceptibility to hypnosis include sending the child to bed or placing him in bed with the implication or verbalization that it is time to be drowsy and to fall asleep, the frequent experience of being told "it doesn't hurt" or "come here and I'll kiss it," and the pat on the head as an application of a soothing agent. Such experiences are significant in creating insensibility and a helpless non-participating compliance, rather than a capacity to react, understand, know and express opinions to the adult. The suggestion to not understand or remember during hypnosis revives previously experienced states of helpless compliance, non-participation, and of infantile and childhood mental limitations. Motor helplessness in the infant and child is recognized by attempting and failing to accomplish an act, or accepting the parental admonitions of "you can't do that" or "you will hurt yourself." This motor helplessness may be revived in hypnosis.

Doctor-Patient Relationship

The transference phenomena seen in psychoanalytic therapy has been used as the basis for a definition and theory of hypnosis by Spiegel: "An altered state of relatedness characterized by irrational submission with abandonment of executive control into a more or less dissociated state, which is actively instigated

and knowingly enhanced by the hypnotist." In the doctor-patient relationship, when hypnosis is used, the patient, in essence, tries to act as a hypnotized person should act in keeping with his own knowledge of hypnosis, and the knowledge which the therapist imparts to him, both focused on a background screen of earlier life experience with authority figures in which many of the features of the hypnotic state have already been experienced.

The possibility of mobilizing such patterns, with their attendant alteration in body chemistry and in motor, sensory, and autonomic nervous system reactions, shows considerable variation from individual to individual in keeping with experiences in the past and with current experiences. The mobilization of the regressive tendencies varies in intensity, depth, and length of time of reaction. The psychotherapist can view his efforts as tending to increase regression, passivity and dependency, on one end of a scale, and tending to encourage independance and self-sufficiency on the opposite end of the scale. A similar scale might be erected as an expression of the patient's desire to regress and to be passive and non-critical, the maximal desire and need to regress being on one end of the scale and minimal tendency on the other end.

Hypnosis as a Therapy: Many therapeutic influences can be placed on a scale of expectations. On one end of the scale no expectations are placed upon the patient (if this is possible). At the opposite end is the expectation of unquestioning submission, and an acceptance of ways of thinking and behaving with the expectation of receiving unlimited care, protection and attention in return. In between these polar opposites are many states of admixture of degrees of no expectation and unlimited expectations. People are constantly exposed to suggestions of all kinds directed at influencing their behavior. The suggestions range from out and out falsifications, as in some advertising, which are none the less accepted and form a basis of action, to the experiences in medical practice which need be no more than exposure on the part of the patient to the statement "you'll feel better." Persuasion through continuous statements of urging, or exhortation to behave in a certain way, is another method of influencing the patient. The development of habits of unquestioning obedience and submissive acquiescence and the establishment of faith in an ideal, a philosophy, a theory, a military establishment, a religion, or a teacher all can be related to that facet in interpersonal relationships in which the role of healer and patient is accepted by each participant. The use of hypnosis, as against other therapeutic endeavors, varies with the possibilities of treatment by other means and the competence of the therapist. Treatment often is "therapist-oriented" and not "patient-oriented." The less the psychotherapeutic skills of the therapist, the more likely will he be to use hypnosis as a therapeutic method (especially if this is the area of his greatest skill and his "God complex" is in need of constant feeding).

Hypnosis as a therapeutic measure would seem to have a place in psychotherapy where: (1) the therapist's skills are primarily in the area of increasing the regressive tendencies on the part of the patient. The therapist in essence prohibits the patient from having symptoms, encourages obedience and helps create an acceptance of a feeling that he is omniscient. The patient needs or desires the regressive position of submission to authority. The treatment situation does not offer too much possibility of more effective help. (2) Acute disturbances affect fairly large numbers of individuals in a similar manner as in war neuroses. The goal of the therapist is alleviation of the immediate symptoms. (3) The patient might be considered for therapy of specific symptoms not readily relieved by drugs, or a rational patient-doctor relationship. Often patients cannot undertake a treatment procedure in which there is a possibility of altering personality structure. A shortage of trained psychotherapists and economic factors decrease the possibility of offering such treatment. (4) The removal of a symptom which helps create a vicious cycle leads thereby to restoration of previous

effective patterns of reacting, as in the case where a well-established balance of a passive receptive dependency with another individual (husband or wife) has been dissolved, there may be a need to re-establish the tendency to repress. The previously passive, dependent wife may be in good balance with a somewhat aggressive husband. A breakdown in this pattern may lead to expressions of hostility toward the husband which in turn creates fear of retaliation. Hypnosis might be used to bring about repression of the hostility, diminish anxiety and prevent a disruption in an interpersonal relationship which is as good as one can expect for that patient. (5) Psychoneurotic disturbances in patients can be improved through the feeling that one is receiving the love, affection, interest, and protection of a person who may substitute for significant figures in the past or present. Hypnosis may create a sense of having someone who cares, and therefore restores the felling that love and security can be expected. This may be temporarily substituted for that which is lacking in the real life situation and enable one to begin psychotherapeutic work.

Rosen has given the following indications for hypnosis as a therapeutic agent: (1) for sedative, analgesic or anesthetic purposes; (2) to make symptoms disappear; (3) for tranquilization (prior to dentistry, surgery, or situations creating anxiety); and (4) as an adjuvant technique in the treatment of neuroses and psychoses. However, there is some opposition to the use of hypnosis in the psychiatric states, especially for paranoia. Hypnosis, when used by **non**-psychiatrists, should be only for purposes related to the particular specialty and competence of the therapist.

Lee has stated that hypnosis may be considered necessary when other more conventional techniques have not been illuminating, or unavailable for special reasons or when there is an inability to reach the patient because of the time factor, education, primipara, chronicity, or age. It is useful in:

1) differential diagnosis between organic and functional symptoms.
2) explanation of the roots of some deeply seated complex
3) weakening the intensity of repressive forces.

Hypnosis, we have found, does not accomplish much that cannot be accomplished by a relationship in which less of the regressive needs of the patient are mobilized. The magical qualities of a psychotherapeutic procedure need not be purposely fortified for most patients seeking treatment.

BIBLIOGRAPHY

1. Bernheim, H.: Suggestive Therapeutics (trans. by C. A. Herter). New York, Putnam, 1900.
2. Braid, James: Neurypnology, or the Rationale of Nervous Sleep. London, 1843.
3. Esdaile, J.: Natural and Mesmeric Healing. Ref.: In Personality and Behavior Disorders, edited by J. McV. Hunt. New York, The Ronald Press, 1945.
4. Forel, A.: Hypnotism: Or Suggestion and Psychotherapy (trans. by H. W. Armit). New York, Rebman, 1907.
5. Freud, Sigmund: The Dynamics of Transference: Collected Papers 11. London, Hogarth Press, 1912.
6. Grote, L. R.: "Die Medizin der Gegenwart in Selbsdarstellungen," vol. 4, pp. 1-51. Leipzig, 1925.
7. Janet, P.: Psychological Healing. New York, Macmillan, 1925.
8. Lee, John C.: Personal Communication.
9. Linder, R. M.: Hypnoanalysis as a Psychotherapeutic Technique, Specialized Techniques in Psychotherapy, pp. 25-39. Grove Press, Inc., New York, 1952.
10. Masserman, Jules: Principles of Dynamic Psychiatry. Philadelphia, Saunders & Co., 1955.
11. Pavlov, I. P.: Lectures on Conditioned Reflexes (trans. by W. J. Grant). Livenwright Pb. Co., 1928.
12. Rosen, Harold: Hypnosis: Application and Misapplication. J.A.M.A., *172*:683, February 13, 1960.
13. Schilder, P. and Kauders, O.: Hypnosis Nervous and Mental Diseases. Monographs Publishing Co., New York, 1927.
14. Schultz, J. H.: Psychotherapie. Stuttgart, 1952.
15. Speigel, Herbert: Hypnosis and Transference. Arch. of General Psych., *1*:634, 1959.
16. Warren, H. C.: Dictionary of Psychology. Boston, Houghton Mifflen, 1934.
17. West, Louis J.: Psycholophysiology of Hypnotism. J.A.M.A., *172*:672, February 13, 1960.
18. White, R. W.: A Preface to the Theory of Hypnotism. J. Abnormal Soc. Psychol., *36*: 447, 1941.
19. Zilboorg, G. and Henry, G. W.: A History of Medical Psychology. Norton, New York, 1941.
20. Zweig, S.: Mental Healers. Cassel, London, 1933.

AMNIOTIC FLUID INFUSION SYNDROME

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The infusion of amniotic fluid into the circulation of the pregnant patient was first described as a clinical entity in 1941 by Steiner and Lushbaugh[1]. They reported a series of six cases examined at necropsy. The first case report was that of Meyer[2] in 1926.

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The reported incidence of this condition varies tremendously. Steiner and Lushbaugh reported an incidence of one case in 8000 deliveries at Chicago Lying-In Hospital in 1941. Sluder and Lock[3] found 4 cases of this condition in 1000 consecutive maternal deaths in North Carolina. A maternal mortality study in 1951 at Charity Hospital, New Orleans, covering a period of 10 years, included no cases of this syndrome[4]. In 1959 a 7 year maternal mortality study in the

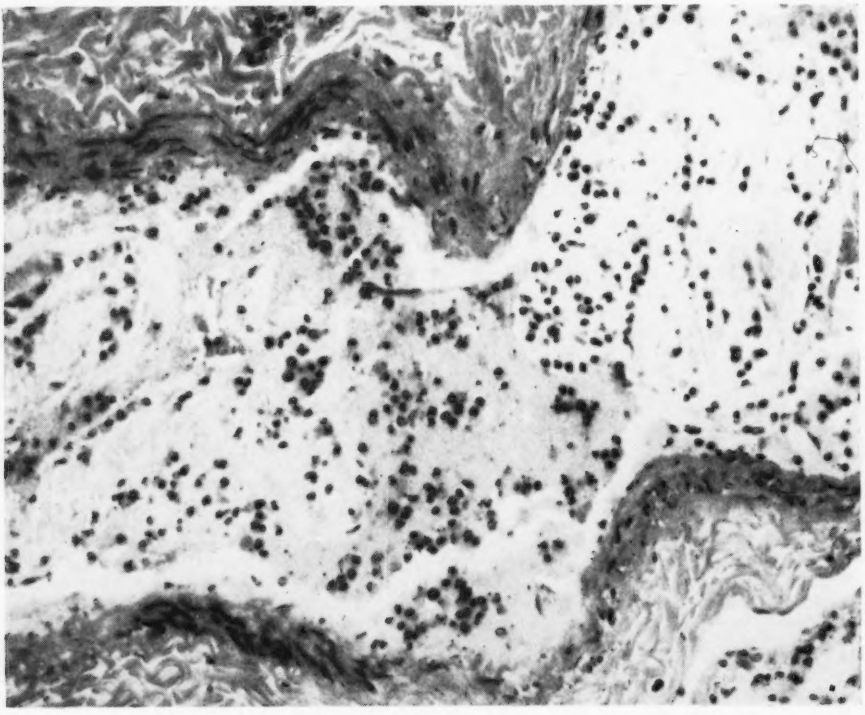

Fig. 1. Pulmonary embolus showing its mucinous component.

State of Minnesota[5], revealed 15 cases of amniotic fluid embolism, an incidence of one case per 37,323 deliveries. In a review of 408 maternal deaths in Ohio in the years of 1955-1958, 11 cases of amniotic fluid infusion were found[6]. About 4 to 5 cases of this entity are reported each year in the foreign literature.

This syndrome usually has an abrupt onset in a pregnant patient at or near term, or post term. All patients have a history of ruptured membranes from a few hours to a few days prior to the onset of symptoms. There is a greater frequency of multiparous patients. More commonly the uterine contractions have been described as intense to tetanic, although apparently normal contractions have been described in a great number of these cases.

The symptoms are restlessness, chill or chilliness, dyspnea, cyanosis and profound shock[1,5,7,9]. Reid et al.[7] describe uterine hemorrhage due to incoagualibility of the blood in 40 per cent of the patients. The duration of symptoms until death varies from a few minutes to several hours.

Pathogenesis

Amniotic fluid gains access to the maternal circulation via the utero-placental vessels or cervical veins and is then carried to the pulmonary circulation. It has been seen in patients with abruptio placentae, at caesarean section, partial placenta accreta, ruptured uterus and more frequently where none of these conditions were present. Many feel that high rupture of the membranes with obstruction by the presenting part to the egress of the fluid permits a separation of the membranes from the uterus and entrance of amniotic fluid into the maternal circulation via damaged utero-placental vessels and sinusoids[1,5,7].

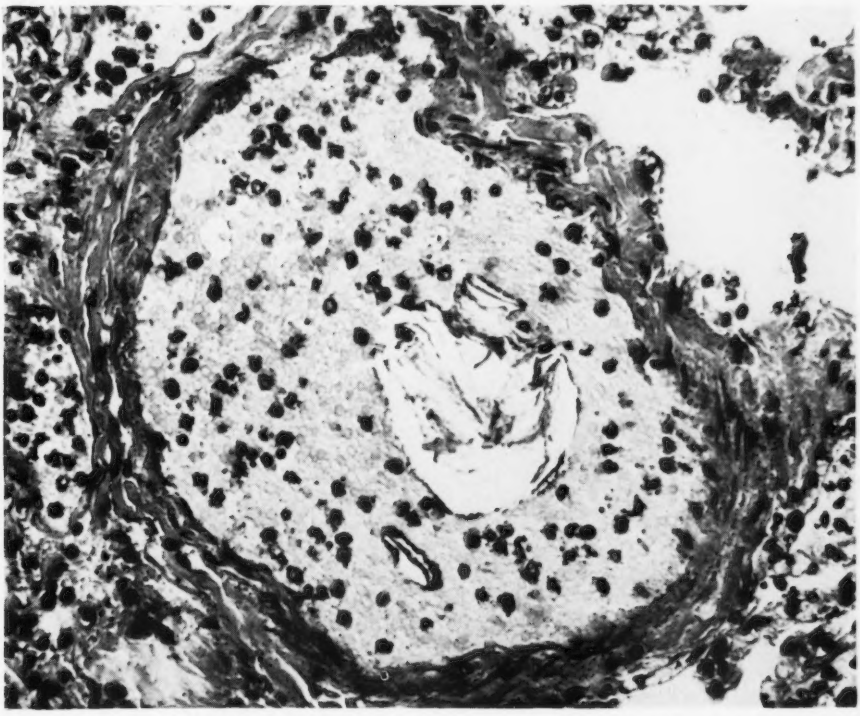

Fig. 2. Pulmonary embolus showing amniotic squames in pulmonary vessel.

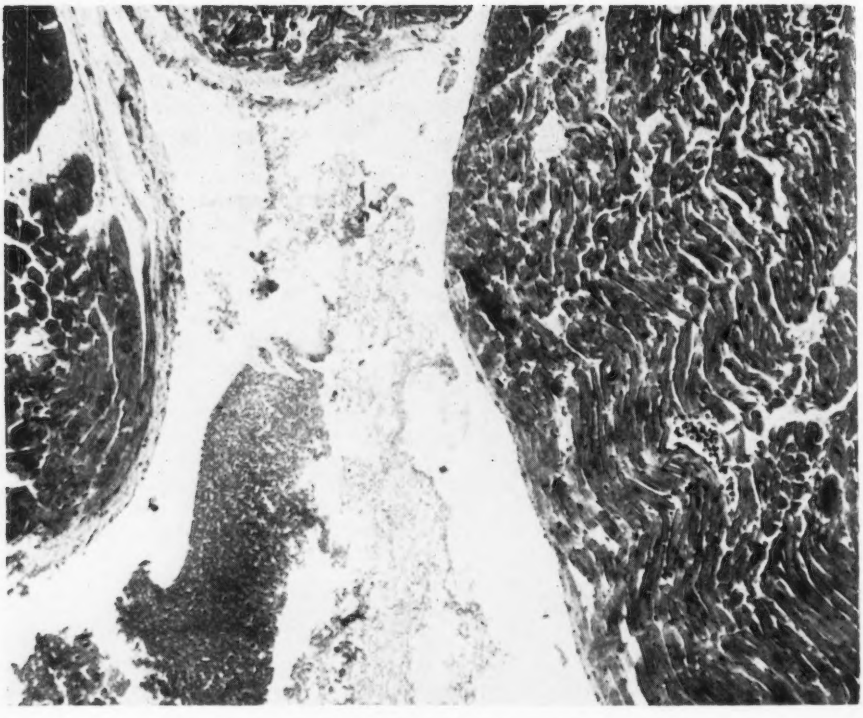

Fig. 3. Amniotic debris in right ventricular cavity.

Several patients in the reported cases received oxytocics during the labor before the catastrophy. Death may occur in just a few minutes and gross examination of the tissues at autopsy may reveal no evidence of the cause of death.

The microscopic findings are dramatic and startling[1,5,7,8]. In the smaller pulmonary vessels throughout the lungs, evidence of amniotic infusion is found, consisting of mucus from meconium-stained fluid, epithelial squames, and lanugo shafts. Where autopsy has not been granted, Gross and Benz[8] described postmortem aspiration of the right side of the heart and the inferior vena cava; they centrifuged the blood and examined the upper layers of the centrifuged blood, which revealed all of the elements described above that have been found in the pulmonary circulation.

Steiner felt that the degree of obstruction of the pulmonary vessels alone could not be the sole cause of death; he postulated that the cause may be an anaphylactoid reaction producing shock and death[9].

Eastman, in an editorial discussion of this subject, quoted the experimental work of Hanzlich and Karsner[10] who, in 1924, reported the results of intravenous injection of small particles of Fuller's earth or charcoal into guinea pigs; they produced symptoms of restlessness, dyspnea, tremors, convulsions, incoagulable blood and death. Marked hemorrhages, congestion, embolism and thrombosis were found in the lungs. They attributed death to an anaphylactoid reaction.

Thromboplastic substances have been isolated from amniotic fluid and Reid et al.[7] described incoaguable blood resulting from the process of intravascular coagulation of this blood with removal of the circulating fibrinogen. In some of these patients fibrinolysins have been isolated,

but the most frequent finding is a hypofibrinogenemia.

Landing[11] reported a study from Boston Lying-In Hospital covering the period 1931 to 1949 of all patients in whom hysterectomy or autopsy followed caesarean section, ruptured uterus or postpartum hemorrhage. He found evidence of amniotic elements in the utero-placental circulation in over 1/4 of these cases. He felt that the syndrome of amniotic infusion did not develop because it only appears when a larger infusion of amniotic fluid occurs. Leary and Hertig[12] demonstrated amniotic fluid elements in 14 placentas studied. They found the amniotic fluid elements between amnion and chorion, in marginal blood clot, in decidua at the placental margin, and in decidual sinusoids on the maternal surface of the placenta.

Tunis[13] reviewed the reports of 25 reported cases of this condition in 1952 and found that 7 patients had internal diseases that might have caused their demise: 6 had rupture of the uterus; 5 had findings insufficient to deny or affirm amniotic fluid embolism. He doubted that condition had been proven as a cause of death.

Therapy

One must be highly suspicious of amniotic fluid infusion syndrome when any of the given symptoms appear. If the condition is suspected before the blood becomes incoagulable, Reid[7] suggests the use of 50 to 75 mgm. of heparin I.V. If, then, hemorrhagic manifestations occur, 4 to 12 grams of fibrinogen are given I.V. along with blood replacement until hemorrhage is controlled. At a later[14] date he reported the survival of 4 pa-

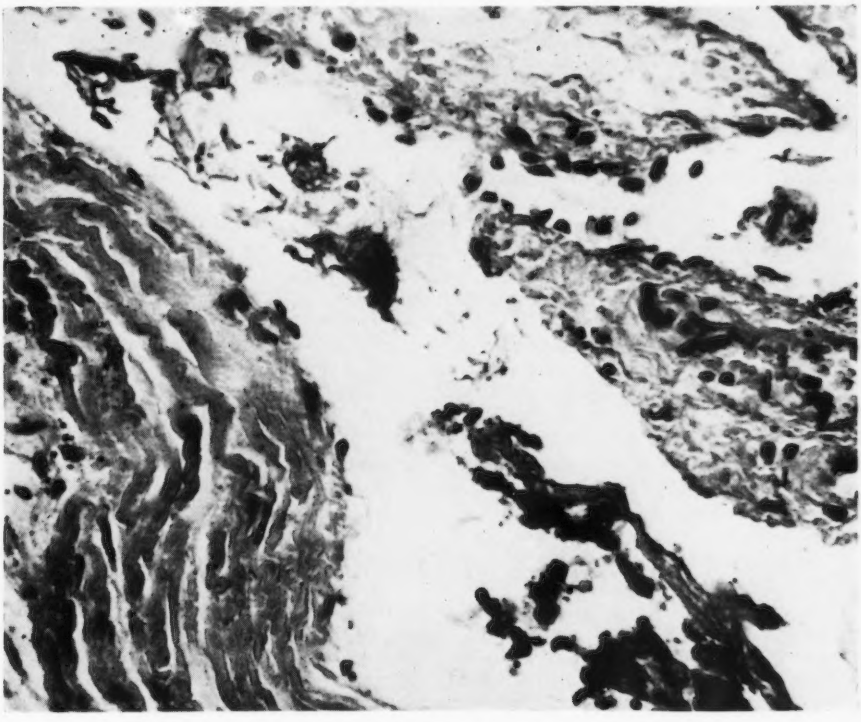

Fig. 4. Amniotic debris and squames in sinusoid of uterus.

tients treated with fibrinogen after a presumptive diagnosis of amniotic fluid embolism. A fifth patient with these symptoms, not given fibrinogen, demonstrated at autopsy the presence of amniotic fluid elements in the lungs. During the course of treatment these patients should have several blood samples drawn to see if they would clot, to determine levels of fibrinogen, and to determine presence or absence of fibrinolysins or other blood clotting factors. Oxygen should be administered because of dyspnea and cyanosis. In order to combat the anaphylactoid reaction Barno and Freeman[5] suggest the use of papaverine and atropine to ward off vagal effects. Others have suggested the use of hydrocortisone and norepinephrine to combat shock and to prove that amniotic fluid infusion occurred.

REFERENCES

1. Steiner, P. E. and Lushbaugh, I. C. C.: J.A.M.A., *117*:1245 and 1340, 1941.
2. Meyer, J. R.: Brazil-Medico., *2*:301, 1926.
3. Sluder, H. M. and Lock, F. R.: Am. J. Obstet. and Gynec., *64*:118, 1952.
4. Kantor, H. J.: Am. J. Obstet. and Gynec., *77*: 1207, 1959.
5. Barno, A. and Freeman, D. W.: Am. J. Obstet. and Gynec., *77*:1199, 1959.
6. Ruppersberg, A., Jr.: Am. J. Obstet. and Gynec., *77*:1209, 1959.
7. Reid, D. E. and Weiner, A. E.: New England J. Med., *243*:597, 1950.
8. Gross, P. and Benz, E. J.: Surg. Gynec. and Obst., *85*:315, 1947.
9. Eastman, N. J.: Obst. and Gynec. Survey, *3*: 37, 1948.
10. Hanzlich, P. J. and Karsner, H. T.: J. Pharm. Exper. Therap., *23*:173, 1924.
11. Landing, B. H.: New England J. Med., *243*: 590, 1950.
12. Leary, O. C., Jr. and Hertig, A. T.: New England J. Med., *243*:88, 1950.
13. Tunis, B.: Am. J. Obst. and Gynec., *64*:72, 1952.
14. Reid, D. E., Weiner, A. E. and Roby, C. C.: Am. J. Obst. and Gynec., *66*:465, 1953.

HEMORRHOIDECTOMY: MINIMIZING POSTOPERATIVE PAIN

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Hemorrhoid surgery has acquired a reputation for having a very disagreeable and painful postoperative recovery period. However, hemorrhoidectomy need not be followed by a great deal of postoperative pain. By paying attention to every detail during surgery as well as in the preoperative and postoperative periods, the surgeon can reduce pain to a minimum. The patient is usually pleasantly surprised by the relative comfort that he enjoys following hemorrhoidectomy and during the subsequent bowel movements. I will outline the methods I employ to achieve the objective of a thorough hemorrhoidectomy followed by a rather comfortable postoperative period.

The Preoperative Period

The surgeon must allay the patient's fears at the time of the original interview. The patient must be assured that there will be only a small amount of discomfort after surgery. In particular, the patient must be told that his postoperative bowel movements will not be accompanied by a great deal of discomfort.

Mineral oil is started about three to five days before surgery. One ounce is taken at bedtime. Another one-half to one ounce is repeated in the morning if the patient is very constipated. On the evening before surgery, enemas are given until the water returns clear. Generally, not more than three or four enemas are permitted even though the return flow is not perfectly clear.

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The Anesthetic

Spinal anesthesia is the anesthetic of choice in most cases. It gives excellent relaxation and exposure to facilitate a meticulous and gentle dissection. Local anesthesia is satisfactory but not ideal. I prefer not to inject anything into such an area of contamination. General anesthesia is best supplemented by local anesthesia and therefore, not generally used as the primary anesthetic for hemorrhoidectomy. If a patient wishes to be asleep during the operation, a spinal anesthetic can be given and followed by intravenous or very light inhalation anesthesia.

The Operation

There are many ways of performing a successful hemorrhoidectomy. The method I prefer involves a suture ligature at the proximal end of each hemorrhoidal mass. This is followed by excision of the internal and external hemorrhoid with its overlying mucous membrane and skin. A sufficient bridge of mucous membrane must be left between each hemorrhoidal mass removed. Extreme care is taken to isolate the sphincter and free it completely from the hemorrhoidal mass so that no part of it is clamped or ligated. The sphincter is not unduly stretched or dilated prior to the hemorrhoidectomy so that none of its fibers is torn. No crushing clamps are placed on any tissue that is not to be excised.

No sutures are placed in the mucous membrane or skin, aside from the original suture ligature at the proximal portion of the hemorrhoid. This practice not only results in adequate drainage and lack of infection but also promotes heal-

ing without stricture formation. When the skin and mucosa are left open, the healing is as rapid as if sutured and is attended with less postoperative pain.

A few words must be stated about the injection of anesthetics in oil in the anal region at the time of surgery. I abandoned this practice many years ago after a thorough trial. The oily solution acts as a foreign body even when deposited in very small amounts in any one area. This foreign body reaction often results in induration and pain as well as delayed healing. In addition, there is always the possibility of a small abscess developing at the site the oil is deposited. In summary, I have found that there is less postoperative pain and more rapid healing when anesthetics in oil are not injected into the area at the time of surgery.

The Postoperative Care

The most acute pain of surgical procedures is usually noted when the anesthetic first wears off. Therefore, I order an intramuscular injection of Demerol or similar narcotic to be given the moment that there is any sensation perceived in the operative area. The object is to prevent any vicious cycle of pain, sphincter spasm, more pain, etc. If the pain is prevented from the start, the postoperative period will be a fairly comfortable one. Therefore, pain medication is given before the pain occurs rather than waiting until the patient is uncomfortable. The patient is given 30 mg. (½ grain) of codeine or its equivalent every four hours for three to five days. This medication is also given at night if the patient awakens. The codeine is often combined with aspirin or empirin compound for greater effectiveness. A slow release, long acting form of codeine (Donagesic Extentabs) can be given at bedtime to obviate any need for medication at night. Any constipating effects of the codeine are overcome by the large amounts of mineral oil given to the patient.

Mineral oil is prescribed postoperatively to insure soft oily stools. The patient is given one ounce of the oil three times daily. It is not desirable to have a bulky stool in the first four or five days after hemorrhoidectomy. Therefore, bulk laxatives such as Metamucil are not prescribed during the early postoperative period. Metamucil is, however, often instituted starting on the fourth or fifth day after surgery. At the same time the mineral oil is gradually reduced from three ounces to one or one and one-half ounces daily in divided doses. No enemas are necessary with the regime outlined above. If the patient has not begun to move his bowels in 72 to 84 hours after surgery, the codeine is reduced or temporarily discontinued in order to allow more active peristalsis.

The patient is given a general diet except for nuts, seeds, and highly seasoned foods. Warm moist compresses to the anal region are regularly employed starting about six hours after surgery. About 36 to 48 hours after surgery, sitz baths are substituted for the compresses. It is especially important to have the patient take a sitz bath following each bowel movement. This results in relief of any discomfort and allows for more adequate cleansing. Between baths, an electric heating pad over the anal region may be used for additional comfort. Wiping of the anal region is done with soft facial quality tissues. Nupercaine ointment is applied externally to the operative area several times daily. The operative area is not traumatized by the insertion of suppositories or other medication into the anal canal.

Digital examinations are seldom necessary in the first 10 to 14 days after hemorrhoidectomy. About two weeks postoperatively, a gentle digital examination is performed and repeated every week for about six weeks. Rarely is any form of "stretching" of the anal canal required. Strictures are not observed when hemorrhoidectomy is performed according to the technique described.

CARDIAC OUTPUT BY BALLISTIC METHODS*

JOHN L. NICKERSON, Ph.D.**

INTRODUCTION

The measurement of cardiac output has been long a challenging problem. Many measurements offer the choice between direct and indirect methods of observation. However, for cardiac output, the direct measurements of the heart's blood ejected into a measuring flask is merely of academic interest so that the measurement of cardiac output is essentially an indirect procedure. Cardiac output is the blood circulated in the systemic circulation. It is measured in liters of blood per minute. Sometimes, for comparison between individuals of different sizes, the measurement is reported as liters of blood per minute per square meter of body surface. This value is called the Cardiac Index. Cardiac stroke volume is the volume of blood ejected from the left side of the heart in cubic centimeters per beat of the heart.

Physical methods which have been discussed in detail in other sources include the Cardiometer or Heart Plethysmograph[1], the Rotameter[2], and the Magnetic Flow Meter[3]. Methods depending upon the Fick principle include the Foreign Gas Method[4,5,6], and the direct Fick with right heart catheterization[7], the injection method devised by Stewart[8], the pulse pressure method improved by Hamilton[9], and the Roentgen Ray Methods[10,11]. The only methods which have not been presented together elsewhere are the methods dependent upon the Ballisto-cardiograph and based upon a non-empirical theoretical approach, and it is these which shall be discussed here.

BALLISTOCARDIOGRAPHIC METHODS

The motions of the body resulting from its recoil to the momentum changes produced in the blood by the beating of the heart were first noticed and reported by Gordon in 1887[12]. These movements of the body were attributed by him to the heart's activity. Later, in 1905, Henderson[13] used a simple suspended platform on which the subject reclined to demonstrate these ballistic movements. Henderson suggested that the magnitude of these movements was related to the amount of blood pumped by the heart.

It was not until the 193?'s that any concentrated study of these phenomena was made. At that time Starr[14] initiated research which has continued to the present time and which has been extended and modified by many workers. Starr, using a simply suspended platform on which the subject lay, stabilized this system by means of a stiff spring mounted so as to control its movements in a longitudinal direction. Because of the spring attachment, the device became a high-frequency instrument. This high-frequency ballistocardiograph produced definite patterns which varied from normality with defects in the heart's action and varied in size with the quantity of blood ejected

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by the heart. Starr[15] also deduced the "force" of the heart at ejection of blood in systole and suggested this measure was of considerable diagnostic importance.

Subsequently in the 1940's, Nickerson[16] and co-workers, theorizing that a less rigidly suspended system would provide more precise information on these ballistic activities, introduced a low-frequency, critically damped system. Workers using this type of ballistocardiograph also were able to demonstrate abnormalities in the recoil patterns due to myocardial damage[17], coarctation of the aorta[18,19], and other defects and a relationship[20] between size of pattern and amount of blood pumped per beat of the heart.

In 1940, Dock[21] suggested the use of a ballistocardiograph in which the patient was supported only by the firmly anchored rigid table upon which he lay. Ballistic movements were recorded directly from the patient's body by means of a rigid bar placed across the shins. This system depends to a very high degree upon the body-spring characteristic of the subject and since one of the natural frequencies of the body lies in the range of three or four cycles/sec., which is also within the frequency spectrum of the ballistocardiogram, distortion of the movement of the body by resonance seriously confuses the pattern. It was also found that this system is not useful for numerical caluclations dependent upon the dimensions of the pattern. However, because of the apparent simplicity of the device, this direct ballistocardiograph has had a considerable clinical vogue.

In 1953, Burger[22] and VonWittern[23], and in 1954, Talbot[24], showed that the low frequency type of instrument was preferable to high-frequency ballistocardiographs. These investigators advocated the use of ballistocardiographs in which the natural frequency was even lower than that used by Nickerson. It appears then that the most satisfactory ballistocardiographs, from theoretical points of view, are those in which the natural frequency of the instrument is of the order of 1/2 cycles per second or less and with damping of the order of the critical value. It must be remembered, however, that under certain circumstances the use of the ultra-low frequency systems is impractical and, for reasons of expediency, the use of middle or high-frequency instruments is forced upon the investigator.

The use of these instruments in the attempt to determine the cardiac stroke volume has been undertaken mainly by three groups of workers. The first group with Starr[14] utilizes the high-frequency ballistocardiograph; the second with Klench[25] uses the ultra-low frequency system; and the third with Nickerson[26,27] bases its calculations upon the characteristics of instruments of the low-frequency, critically damped type.

Many methods of recording the movement of the ballistic system have been used. Ballistocardiograms may be classified as displacement, velocity or acceleration records, depending upon which function is actually recorded. However because of the coupling between body and ballistic systems, these records are not identical from one type of ballistocardiograph to another. For example, the displacement pattern recorded on a Starr instrument resembles in appearance, not the displacement record of the ultra-low frequency system, but rather the acceleration pattern obtained from this system.

The basic theory for the methods of computing cardiac stroke volume is described in the following paragraphs:

The recoil of the body to the heart's ejection is governed by Newton's law of conservation of momentum. The application of the law in this case may be stated as follows:

$$M_b V_b = m_r v_r + m_l v_l \qquad (I)$$

where M_b, V_b, m_r, v_r, and m_l, v_l are mass and velocity of bed and body, and mass and mean velocity of blood ejected from the right and from the left sides of the heart. In these methods as used at the present time, it is assumed that the mass and the mean velocity of blood ejected from both sides of the heart in the normal subject are equal. It therefore becomes possible to simplify this equation to the form where the stroke volume (S) is given by

$$S = \frac{M_b V_b}{2dv} \qquad \text{(II)}$$

where "d" is the density of blood and "v" is the mean velocity of ejection from the heart.

A. Computation According to Starr:

In Starr's formulation, the mean velocity of the blood is given by:

$$v = \frac{S}{AT} \qquad \text{(III)}$$

where "A" equals cross-sectional area of the aorta and "T" is duration of systole. The formula for stroke volume then becomes:

$$S = \sqrt{\frac{M_b V_b AT}{2d}} \qquad \text{(IV)}$$

The duration of systole is related to the length (C seconds) of the heart's cycle by the formula:

$$T = 0.34\sqrt{C}\ \text{Sec.} \qquad \text{(V)}$$

The quantity $M_b V_b$ is derived from the ballistocardiographic record. In the case of Starr's high-frequency system, this quantity is obtained by measuring the area under certain waves in the ballistocardiographic pattern. These areas are under the I-wave and under the J-wave. The areas are measured in units of millimeter-seconds when on the record the movement of the bed under an impressed force of 280 grams is 10 millimeters (See Figure 1).

In final form, the equation for stroke volume becomes:

$$S = 33\sqrt{(2\int Idt + \int Jdt)\,A\sqrt{C}}\ \text{milliliters.} \qquad \text{(VI)}$$

B. Computation According to Klench:

The method of estimating stroke volume used by Klench and co-workers depends also on Newton's law of conservation in the form:

$$mx = My \qquad \text{(VII)}$$

when "m" is the mass of blood moved, "x" is the distance moved by its center of gravity, "M" is the mass of the body and bed and "y" is the distance moved by the center of gravity of the combination of body and bed. Assuming a blood density of 1.06, the stroke volume S becomes:

$$S = \frac{My}{1.06\,x} \qquad \text{(VIII)}$$

From figure 2 one obtains the relationships:

$$Y = \frac{(A)(0.1)}{E}\text{mm} \qquad \text{(IX)}$$

which describes the movement of the center of gravity of the bed and body system from the instant corresponding to point "H" on the ballistocardiogram, when the blood is all within the heart,

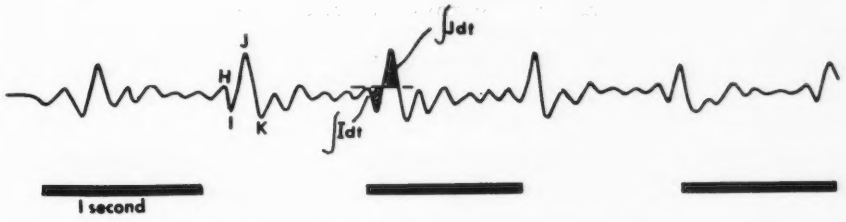

Fig. 1. High-frequency Ballistocardiogram of the Starr type.

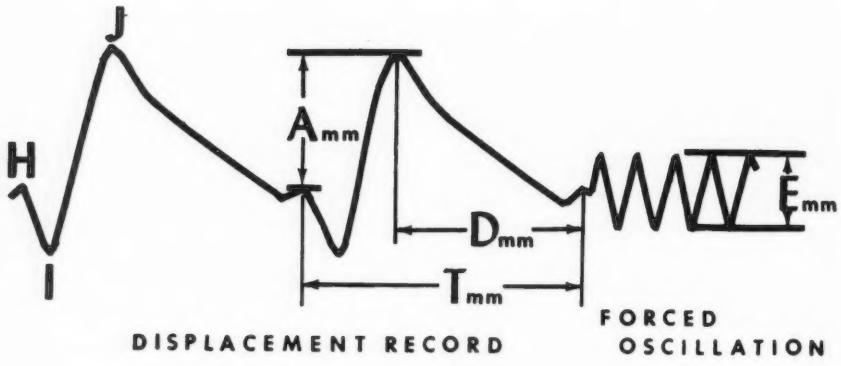

Fig. 2. Ultra-low frequency Ballistocardiogram.

to the time corresponding to point "J." when it is all in the aortic tree. Hence:

$$S = \frac{MA\ 0.1}{1.06\ E\ x} \qquad (X)$$

From trial on models and cadavers, the distance of movement of the center of gravity of the blood between the situations corresponding to "H" and "J" of the ballistocardiogram is given by:

$$L = \frac{120\ H}{170} mm \qquad (XI)$$

when "H" is the height of the subject in centimeters.

The only factor further to consider is that during the period "H-J" a certain run-off of blood from the area has occurred. Assuming that the run-off is of constant rate throughout the cardiac cycle the blood remaining for consideration is the fraction $\frac{D}{T}$ of the whole blood ejected. Therefore, the actual movement of the center of gravity "x" is related to "L" by:

$$\frac{X}{L} = \frac{D}{T} \qquad (XII)$$

Hence, the equation for stroke volume becomes:

$$S = \frac{MA\ (0.1)\ T}{1.06\ E\ LD}\ cm^3 \qquad (XIII)$$

In this formula, "M" is the mass of subject and bed in grams, 0.1 mm is the amplitude of the forced oscillation used in calibration, 1.06 is the density of blood in grams per cubic centimeter, L (mm) is computed from the empirical formula for movement of the center of gravity, and the quantities "A,D,E" and "T" are measured in millimeters on the ballistocardiograph record.

C. **Computation According to Nickerson:**

In the derivation by Nickerson, the approach was to use the equation of motion for the low-frequency, critically damped ballistocardiographic system, namely:

$$M\ddot{x} + b\dot{x} + kx = 0 \qquad (XIV)$$

where "M" is the total mass of body and ballistic bed and where:

$$k = M\omega^2$$
$$b = 2M\omega \qquad (XV)$$
$$\omega = 2\pi f_o$$

f_o being the natural frequency of the ballistic system when undamped.

The general solution of this equation is the formula:

$$X = (At + B)\ e^{-\omega t}. \qquad (XVI)$$

Consider now the events occurring during systolic ejection. At time zero, the blood is ejected headward so that by conservation of momentum, the ballistic system receives a footward impulse giving it a footward velocity, U, and producing the downward sweep of the I-wave. The subsequent displacement of the ballistic system can be described by:

$$X_1 = -Ut_1 e^{-\omega t_1} \qquad \text{(XVII)}$$

During the progress of this footward movement, the blood which has been moving headward decelerates in the pulmonary and head regions and reverses, at the aortic arch, its flow of direction relative to the long axis of the body. These actions produce a headward impulse of magnitude CU, somewhat greater than the original footward impulse. In estimating the factor "C", it was considered that the fraction of blood ejected from the left ventricle which continues headward is "r". The fraction turning the aortic arch is therefore "1-r". The latter fraction gives a double contribution to the headward momentum because of the 180° reversal of direction. Therefore, the factor "C" can be computed from:

(XVIII)

$$C = \frac{[MU + rMU + 2(1-r)]MU}{2MU} = \frac{3-r}{2}$$

For values of "r" between 1/5 and 1/3, the value of "C" = 1.37 ± 3%. Therefore, "C" is taken to be 1.37. The headward movement resulting from the impulse CMU produces the J-wave of the ballistocardiogram. In general this headward movement is terminated by a footward impulse due to blood descending the long aorta and decelerating in its lower regions. If this time of termination is t_2 seconds after time t_1, then the headward displacement is given by:

(XIX)

$$X_2 = e^{-\omega t_2}\left[Ut_2\left(c - e^{-\omega t_1}\right) - Ut_1 e^{-\omega t_1}\right]$$

The total displacement from the trough of "I" to the peak of "J" of the ballistic movement is given by:

(XX)

$$D = X_2 - X_1 = (Ut_2)\left(e^{-\omega t_2}\right)\left(C - e^{-\omega t_1} + \left(\frac{t_1}{t_2}\right)\left(\frac{e^{-\omega t_1}}{e^{-\omega t_2}}\right)\left(1 - e^{-\omega t_2}\right)\right)$$

However, "D" is related to the force "F" grams required to move the ballistic system through this distance according to the formula:

$$980\,F = \omega^2 MD \qquad \text{(XXI)}$$

Therefore, equation (II) for stroke volume becomes:

(XXII)

$$S = \frac{M_b V_b}{2vd} = \frac{MU}{2vd} = \frac{980\,F}{2\omega^2 t_2 Rvd} = \frac{5.27\,F}{t_2 Rv}$$

(XXIII)

$$R = \left(e^{-\omega t_2} \right) \left[C - e^{-\omega t_1} + \left(\frac{t_1}{t_2} \right) \left(\frac{e^{-\omega t_1}}{e^{-\omega t_2}} \right) \left(1 - e^{-\omega t_2} \right) \right]$$

The density of blood being taken as 1.055 and f_o is 1.5

In an earlier empirical expression, no attempt was made to estimate "v", the mean velocity at ejection. It was merely assumed that it was an inverse function of the average blood pressure opposing the ejection of blood. The formula then became, when adjusted against calibration by the direct Fick method[26]:

$$S = \frac{5.02 \ F \ p_a}{R \ T_2 \ H} \text{ milliliters} \qquad \text{(XXIV)}$$

In order to make the computation of stroke volume independent of correlation against another method, direct evaluation of the quantity "v" was attempted. The procedure was to assume that the time between the beginning of footward movement of the ballistic system producing the I wave and the beginning of the footward movement producing the JK-segment is the time for the blood to travel from the heart to some point down the descending aorta. If the total possible distance for this movement is one-fifth of the height of the subject, then a reasonable distance for travel before these effects become apparent is one-half the distance that is one-tenth of the height of the subject. In considering this evaluation, it is likely that in a completely empty aorta, the distance to travel would be the full one-fifth of the height of the subject. Correspondingly, in a completely full and inelastic aorta, the distance of travel before the effects of impact became apparent in the far regions of the aorta, would be zero. This is obvious since the column of blood would move as a whole. Therefore, an estimate of one-half of the distance would be unreasonable in a normal healthy subject. These considerations are not inconsistent with other findings on fluid movements in the aortic tree. Hamilton[28] has shown that the rise of pressure at the beginning of systolic ejection at a point twenty centimeters along the aorta lags about .20 seconds behind the pressure rise of the root of the aorta. For a parabolic wave front this gives a mean velocity of fluid movement of

$$\frac{1}{2} \times \frac{20}{0.2} = 50 \text{ cm/sec.}$$

Pressure equilibrium maximum is reached simultaneously at a point 10, 20 and 30 cm from beginning of aorta at about 0.30 seconds after beginning of ejection. This means that the ejected volume has reached and filled the distal portion of the aorta. The mean distance traveled under these circumstances is about 50 cm/sec. x 0.30 sec., which equals 15 cms. This is about half of the length of the aorta being considered. Hence, our estimate of a practical factor of one-half is not unreasonable.

If now the time for this travel is "T" seconds, then the maximum velocity is $\frac{H}{10\,T}$ and the mean velocity, assuming a parabolic ejection front, is $v = \frac{H}{20T}$. From these considerations, the formula[17] for stroke volume becomes:

$$S = \frac{105\,FT}{RHT_2} \text{c.c.} \qquad \text{(XXV)}$$

In using this equation to estimate stroke volume, the following quantities are measured, some of these quantities being shown in figure 3. The force "F" grams is determined by the proportionality of the distance from the I-trough to the J-peak to the amount of movement caused by the application of a definite force, in this case 50 grams, to the ballistic system in its direction of move-

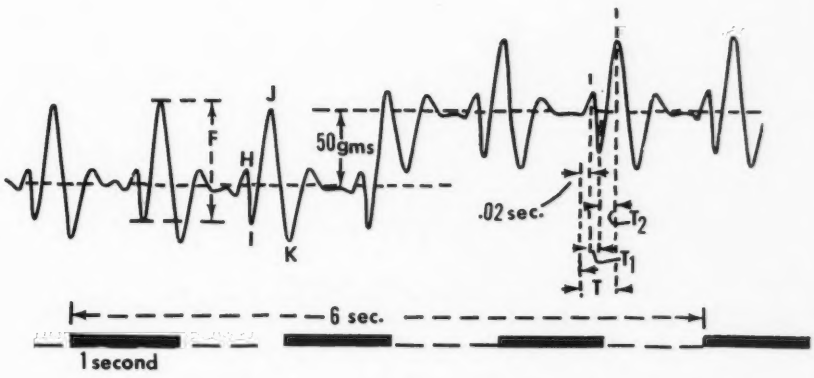

Fig. 3. Low-frequency, critically damped Ballistocardiogram.

ment. T_2 is the time interval in seconds from the I-trough to the J-peak. T_1 is the time in seconds from the H-peak to the I-trough. "T" is the sum of $(T_1+T_2+0.02)$ seconds. The 0.02 seconds is an approximation of the time from the beginning of ventricular ejection to the H-peak[18]. The quantity "R" is determined from figure 4, using the values of T_1 and T_2. "H" is, of course, the height of the subject in centimeters. Insertion of these quantities in the proper units as stated enables one to estimate the stroke volume in milliliters corresponding to any particular ballistic beat. It is obvious that in the derivation of this formula for stroke volume there are many assumptions which are probably only correct to a first approximation. Further investigation of these assumptions is of course indicated. However, since reasonably good agreement has been shown between the stroke volume computed by this formula and the stroke volume estimated from Fick measurements of cardiac output, the method is useful in cases where minimal disturbance of a subject is essential.

The preceding discussions have been concerned with three different types of ballistocardiographs. For each of these instruments and by somewhat different arguments, formulas have been derived which permit computation of cardiac stroke volume. These formulas are not empirical, but are completely independent of other methods for determining stroke volume.

The method by Starr depends on the assumption that the area under the ballistocardiographic curve taken with the high-frequency instrument represents momentum. The method by Klench depends on the view that the displacement traced on the ultra-low frequency ballistocardiographic record represents the displacement of the center of gravity of the blood in the body during the cardiac cycle. The method by Nickerson assumes that the dynamical equation for the low-frequency, critically damped system describes the movement of the ballistic system caused the momentum changes resulting from the motion of blood during the cardiac cycle.

That the results of these computations agree quite well with the results obtained by the Grollman gas or the direct Fick method, indicates that the basic arguments are not seriously in error and that losses in transmission from the heart to the bed are not excessive. Further investigation is necessary to elucidate many points in these derivations. However, for many practical reasons and depending upon the specific situations involved, one or other of these methods can be of value in the non-empirical determination of cardiac stroke volume.

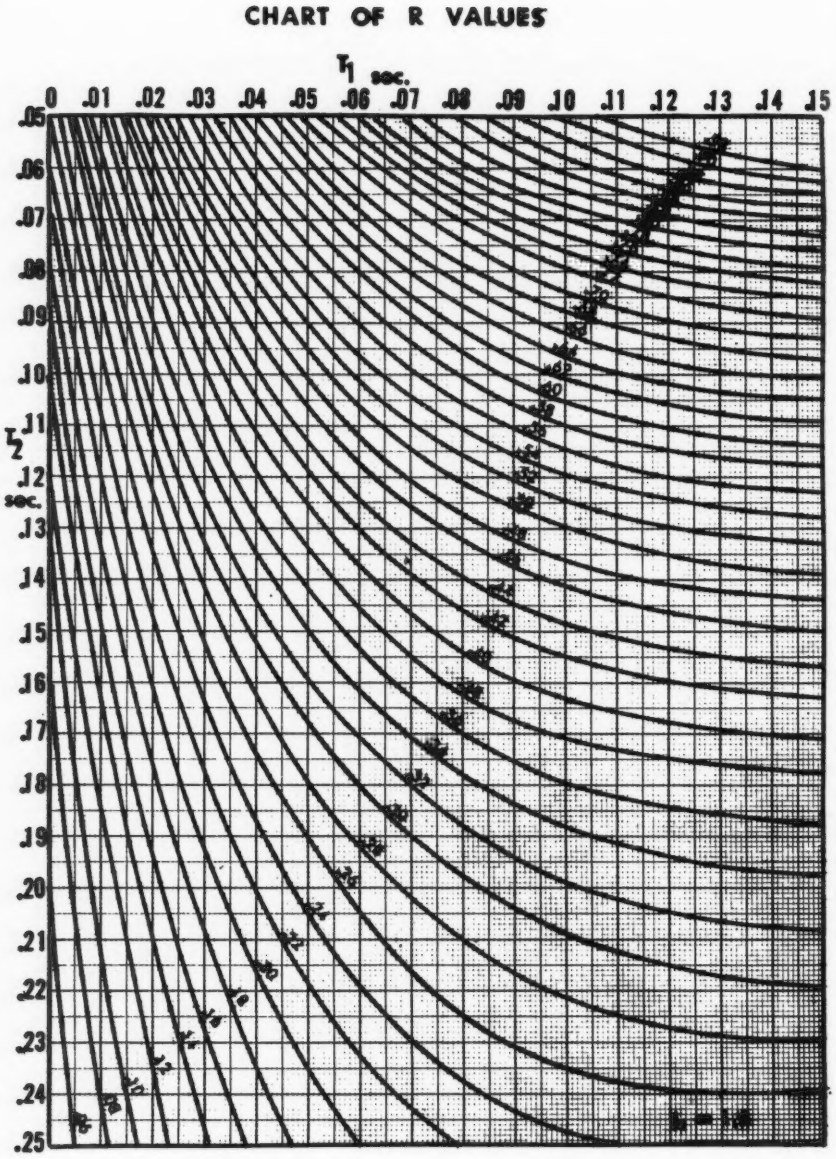

Fig. 4. Table of R-values for 1.5 cycle critically damped Ballistocardiogram.

BIBLIOGRAPHY

1. Henderson, Y., Scarborough, M. M., and Chillingworth, F. P., "Volume Curve of the Ventricles of the Mammalian Heart and Significance of this Curve in Respect to Mechanisms of Heart Beat and Filling of the Ventricles." Am. J. Physiol., *16*, 325, 1906.
2. Longino, F. H., and Gregg, D. E., "Comparison of Cardiac Stroke Volume as determined by the Pressure Pulse Contour Method and by a Direct Method Using a Rotameter." Am. J. Physiol., *167*, 721, 1951.
3. Kolin, A., "An Electromagnetic Flow Meter; Principle of Method and its Application to Blood Flow Measurements". Proc. Soc. Exper. Biol. & Med., *35*, 53, 1936.
4. Krogh, A., and Linhard, J., "Measurement of the Blood Flow Through the Lungs of Man". Skandinav. Arch. f Physiol., *27*, 100, 1912.
5. Grollman, A., "The Cardiac Output in Man in Health and Disease." Publisher C. C. Thomas, Baltimore, 1932.
6. Starr, I. Jr., Donal, J. S., Margolies, A., Shaw, R., Collins, L. H., and Gamble, C. J., "Studies of the Heart and Circulation in Disease; Estimation of Basal Cardiac Output, Metabolism, Heart Size and Blood Pressure in 235 Subjects". J. Clin. Invest, *13*, 561, 1934.
7. Cournand, A., "Measurement of Cardiac Output in Man Using Right Heart Catheterization". Fed. Proc., *4*, 207, 1945.
8. Stewart, G. N., "Output of the Heart in Dogs". Am. J. Physiol., *57*, 27, 1921.
9. Hamilton, W. F., and Remington, J. W., "Measurement of the Stroke Volume from the Pressure Pulse". Am. J. Physiol., *148*, 14, 1947.
10. Eyster, J. A. E., and Meek, W. J., "Instantaneous Radiographs of the Human Heart at Determined points in the Cardiac Cycle". Am. J. Roentgenology, *7*, 471, 1920.
11. Johnson, S. E., "Roentgen Kymography Considered in Relation to Heart Output and a New Heart Index". Am. J. Roentgenology, *37*, 167, 1937.
12. Gordon, J. W., "On Certain Molar Movements of the Human Body Produced by the Circulation of the Blood". J. Anat. Physiol., *11*, 533, 1877.
13. Henderson, Y., "Mass Movements of the Circulation as Shown by a Recoil Curve". Am. J. Physiol., *14*, 287, 1905.
14. Starr, I. Jr., Rawson, A., Schroeder, H. A., and Joseph, N. R., "Studies on the Estimation of Cardiac Output in Man and Abnormalities in Cardiac Function from the Heart's Recoil and the Blood's Impacts; the Ballistocardiogram". Am. J. Physiol., *127*, 1, 1939.
15. Starr, I., "Normal Standards for Amplitude of Ballistocardiograms Calibrated by Force". Circulation, *11*, 914, 1955.
16. Nickerson, J. L., and Curtis, H. J., "The Design of the Ballistocardiograph". Am. J. Physiol., *142*, 1, 1944.
17. Mathers, J. A. L., Nickerson, J. L., Fleming, T. C., and Patterson, M. C., "Abnormal Ballistocardiographic Patterns in Cardiovascular Disease as Recorded with Low-Frequency, Critically Damped Ballistocardiograph". Am. Ht. Journal, *40*, 390, 1950.
18. Nickerson, J. L., "Some Observations on the Ballistocardiographic Pattern, with Special Reference to the H and K Waves". J. Clin. Invest., *28*, 369, 1949.
19. Nickerson, J. L., Humphries, G. H., Deterling, R. A., Fleming, T. C., and Mathers, J. A. L., "Diagnosis of Coarctation of the Aorta with the Aid of the Low-Frequency, Critically Damped Ballistocardiograph". Circulation, *1*, 1032, 1950.
20. Nickerson, J. L., Warren, J. V., and Brannon, E. S., "Cardiac Output in Man; Studies with the Low-Frequency, Critically Damped Ballistocardiograph and the Method of Right Atrial Catheterization". J. Clin. Invest., *26*, 1, 1947.
21. Dock, W., and Taubman, F., "Some Techniques for Recording the Ballistocardiogram Directly from the Body." Circulation, *7*, 503, 1953.
22. Burger, H. C., Noordergraaf, A., and Verhagen, A. M. W., "Physical Basis of the Low-Frequency Ballistocardiograph". Am. Ht. Journal, *46*, 71, 1953.
23. Von Wittern, W., "Ballistocardiography with Elimination of Vibration Properties of the Body". Am. Ht. Journal, *64*, 705, 1953.
24. Talbot, S. A., Deuchar, D. C., Davis, F. W. Jr. and Scarborough, W. R., "The Aperiodic Ballistocardiograph". Bull. Johns Hopkins Hosp., *94*, 27, 1954.
25. Klench, H., and Eger, W., "Ein Neues Verfahren der Physikalischen Schlagvolumenbestimmung (Quantitative Ballistographie)". Pfluger's Archivs fur der gesamte Physiologie, *263*, 459, 1956-57.
26. Nickerson, J. L., "The Low-Frequency, Critically Damped Ballistocardiograph". Fed. Proc., *4*, 201, 1945.
27. Nickerson, J. L., "Estimation of Stroke Volume by Means of the Ballistocardiograph". Am. J. Cardiol., *2*, 624, 1958.
28. Hamilton, W. F., "Textbook of Human Physiology." Publisher F. A. Davies Co., Philadelphia, 1949.

VILLOUS TUMOR OF THE RECTUM ASSOCIATED WITH PROFOUND ELECTROLYTE DISTURBANCES

S. LLOYD TEITELMAN, M.D.*

Villous papillomas of the large intestine have become the subject of renewed interest because of the frequency of serious symptoms, and even death, caused by electrolyte depletion. Such lesions necessitate rapid and aggressive diagnosis and treatment.

Papillomas make up 2 to 14 per cent of colonic polyps, occur mostly in the rectum, are in an older age group than adenomas, and have a high malignancy potential.

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Fig. 1. Gross specimen showing bulky, cauliflower-like appearance of tumor.

They are characterized by mucus discharge (pseudo-diarrhea), bloody stools, change in bowel habit and prolapse of the tumor, which is soft and can be passed over easily by the proctoscope. The mucus discharge results from a tremendous increase in secreting surface, with huge numbers of goblet cells, and is high in salt content. This profuse discharge may lead to inanition, dehydration, electrolyte depletion, and azotemia due to the effect of reduced blood volume on renal blood flow.

Four reports in the literature of the past few years deal with this condition, and there are two documented instances of death resulting from it, although others have undoubtedly occurred. We were reminded of a case fulfilling these criteria which came under our care before there was a general awareness of this symptomatology, and felt it would be interesting to re-examine it in the light of present knowledge.

Report of Case

A 55 year old white female was admitted to Michael Reese Hospital on March 9, 1949, with complaints of watery diarrhea (six stools daily), nausea, thirst and weight loss for 12 days; belching and hiccough for 7 days; and oliguria for 2 days.

On physical examination, she was found to be poorly nourished, extremely dehydrated and cachectic. The skin and tongue were dry, and tissue turgor was poor. She was weak, listless and semi-stuporous. Heart tones were feeble and blood pressure was 90/60 mm. Hg. On digital examination of the rectum, a large, cauliflower-like, soft mass was palpated.

Examination of the blood showed hemoconcentration, marked azotemia, and marked hypochloremic acidosis. Hemoglobin was 16.5 grams, hematocrit 49, red blood count 5.3 million, white blood count 32,000. The serum urea

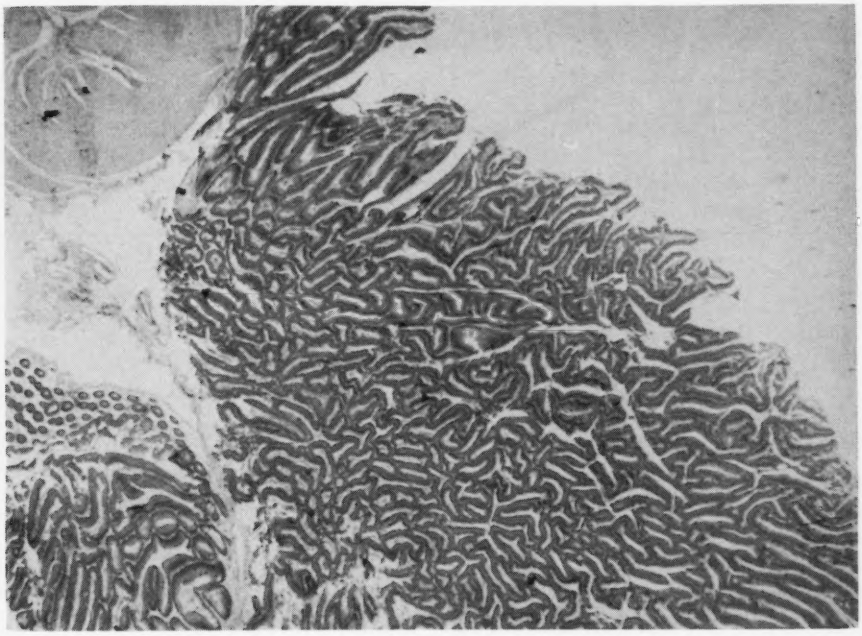

Fig. 2. Low power photomicrograph showing extensive arborization and tremendous secreting surface of tumor.

Fig. 3. Higher power photomicrograph showing details of cellular structure of tumor.

nitrogen was 225 mg. per cent, creatinine 7.6 mg. per cent, chlorides 67.3 milliequivalents per liter and carbon dioxide content 9.6 milliequivalents per liter. Serum total proteins were 7.1 grams per cent with an albumin/globulin ratio of 4.2/2.9.

Vigorous replacement of water and electrolytes was begun, and after forty-

Table 1

		3/10	3/11	3/12	3/14	3/16	3/31	4/7
UreaN	mg.%	225.0	195.0	135.0	60.0	20.5	14.6	11.1
Creat	mg.%	7.6	5.0	2.5	1.0	0.8	0.8	0.7
Cl	mEq/L	67.3	83.3	95.4	103.0	100.0	103.5	
CO_2	mEq/L	9.6	17.3	26.0	23.7	30.3	31.6	
Ca	mg.%		8.8			8.3	10.5	
P	mg.%		2.2			1.4	3.2	
TP	gm.%	7.1		5.0	4.8	5.6	5.0	5.9
Hb	gm	16.5			14.2		9.7	12.7
RBC	mill.	5.3			4.5		3.1	4.1
WBC	thous.	32.1			12.3			11.1
Hct		49.0		41.0	41.0			38.0

Pertinent laboratory determinations showing rapid and dramatic response to therapy.

eight hours the patient was responsive, drinking and urinating, and skin turgor was good. However, diarrhea, weakness, lethargy and nausea persisted.

At this time, twelve years ago, routine determinations of sodium and potassium levels in the serum were not being done. Nevertheless, empirically, a potassium deficit was postulated and the patient was given oral enteric-coated potassium chloride, in daily doses of sixteen grams.

Clinical improvement was complete after four days of replacement therapy. Chemically, the hypochloremic acidosis and hemoconcentration were corrected at the same time. The serum urea nitrogen required an extra day to drop to normal levels. (These values are tabulated in Table 1.)

On the fourth hospital day, proctoscopy was performed. A large, red, granular, friable polypoid lesion was seen on the posterior rectal wall about ten centimeters from the anus. Biopsy was done, and reported as papillary adenocarcinoma.

After two weeks hospitalization, including preoperative bowel preparation, a combined abdomino-perineal resection of the rectum was performed. Figures 1, 2 and 3 demonstrate the gross and microscopic appearance of the tumor, which had dimensions of 8.5 x 7.5 x 1.5 centimeters.

The patient had an uneventful postoperative course, and was discharged from the hospital on the seventeenth postoperative day. At the present time, twelve years later, she is alive and well, with no evidence of recurrent disease.

In conclusion, villous papillary tumors of the large intestine are sometimes associated with profuse mucus discharge which may result in profound electrolyte depletion. Prompt diagnosis and vigorous therapy, consisting of replacement of water and electrolytes, and surgical extirpation of the lesion as soon as the patient's condition permits, will prevent serious consequences, and even death.

BIBLIOGRAPHY

1. Cooling, C. and Marrack, D.: Potassium-secreting Tumor of Colon. Proc. Roy. Soc. Med., *50*: 272, 1957.
2. Goldgraber, M. B. and Kirsner, J. B.: Papilloma of the Large Intestine: A Clinical-Pathological Correlation. Gastroenterology, *35*:36, 1958.
3. McKittrick, L. S. and Wheelock, F. C., Jr.: Carcinoma of the Colon. p. 61, Chas. C. Thomas, Springfield, 1954.
4. Rudman, I., Otto, C. and Novota, O. J.: Villous Papilloma of the Rectum Causing Massive Electrolyte Losses. Arch. Surg., *81*:140, 1960.

DIFFERENTIATION OF ENTEROCOCCI BY THE TETRAZOLIUM REDUCTION TEST

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The group of Enterococci (Lancefield group D) comprise organisms which are biologically active but have a wide metabolic gradient on various substrates. If any one of these characteristics was sufficiently constant, it might be of value in the differentiation of certain species. Not long ago, Laxminarayana and Iya[1] found that faecal streptococci varied in their ability to reduce tetrazolium (TTC). Subsequently, Barnes[2] demonstrated that Streptococcus faecalis can be distinguished from Streptococcus faecium (Orla-Jensen) and other Lancefield group D streptococci by the tretrazolium test. Similarly, by the same method, Dias and da Silva[3] were also able to differentiate between Listeria monocytogenes and Erysipelothrix rhusiopathiae.

In our former blood culture studies[4] Enterococci were recovered from the blood of children with rheumatic fever. Hence, the question on hand was whether or not in this disease one is dealing with a homogenous or non-homogenous group of organisms.

Methods

The procedure adopted in handling the culture and in performing the test was essentially the same as the one described by Barnes with only minor changes to suit our conditions. The formazen dye was extracted with n-butanol from the cultures and the color intensity measured in a Coleman Junior Spectrophotometer at 500 mu, using control tubes without organisms as reference.

The strains to be tested (32) have been described in detail in our previous publication[4]. They all met the biological and serological requirements of group D Enterococci. As reference the following strains from the ATCC were used: two cultures of Strep. durans (6056, 9810); four of Strep. faecalis (828, 4082, 4200, 4835); two of Strep. liquefaciens (4532, 4533); and two of Strep. zymogenes (6054, 6641).

Otherwise the test was thoroughly standardized: the culture medium was adjusted to an initial pH of 6.6; the incubation period was uniformly 18 hrs. at 37° C.

Results

Table I contains the pertinent data of our experiments.

Examining the reference strains first, it seems evident that at least three of the four Sherman groups[5] can be distinguished by the degree of their tetrazolium reducing capacity. These three groups are quite distinct with an average reduction of 2.7% for Strep. durans; 55.0% for Strep. faecalis and about 75.0%

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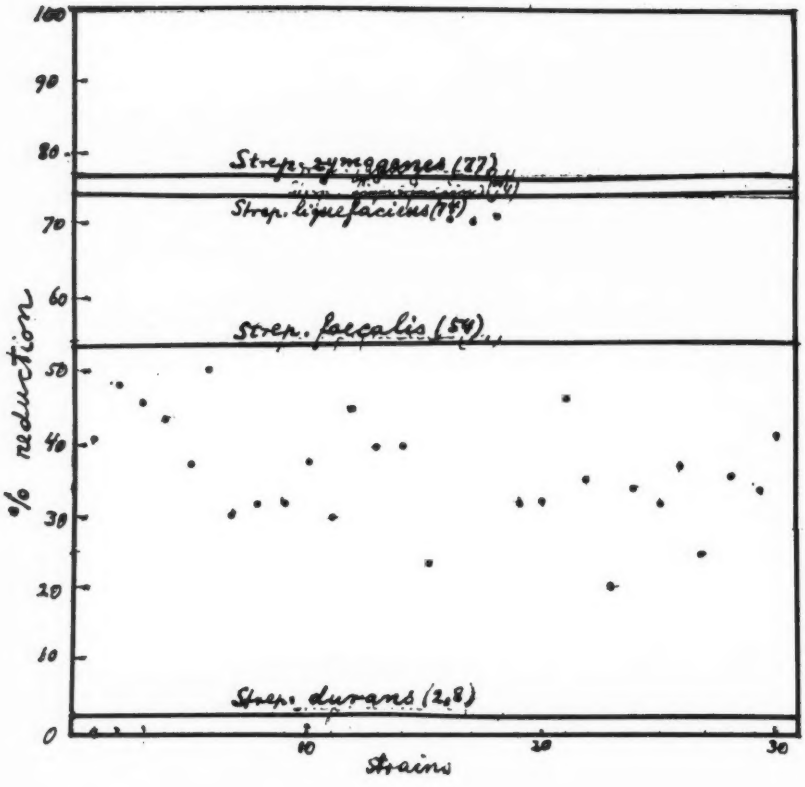
Table I

The reduction of tetrazolium by Enterococci (Group D)

a) freshly isolated strains from blood cultures b) reference strains (ATCC)

Origin of strains	# of strains and % reduction to forazan									
	1		2		3		4		5	
	#	%	#	%	#	%	#	%	#	%
a	3[1]	41	5	44	24	40	43	38	31	50
	3[2]	48	6	37	26	31	47	35	32	55
	3[3]	46	15	31	27	40	48	21	33	56
	8[1]	30	16	34	29	24	49	34	42	53
	8[2]	31	17	38	37	32	51	32		
	13[1]	36	19	30	38	32	52	37		
	13[2]	33	23	45	39	46	53	25		
b	Strep. durans		6056	3.0	Strep. Liquefaciens		4532	74.5		
			9810	2.5			4533	73.0		
	Strep. faecalis		828	55.0	Strep. zymogenes		6054	76.0		
			4082	58.0			6641	78.0		
			4200	55.5						
			4835	52.0						

Indices in column 1 mean repeated isolations from the same patient



for the remaining two groups (Strep. liquefaciens and Strep. zymogenes). An additional number of authenticated strains would greatly implement this characteristic difference. However, the good agreement among the few strains presently available will serve well for comparative purposes.

Analyzing test strains isolated from the blood of patients (32) one gains on first glance the impression that there is a rather wide range of scatter among these strains (21-56%). On closer observation, however, one can see that the majority of strains (28) listed in the first 4 columns form a distinct group with reducing activities falling between 21-48% and an average of 35.4%. Only four strains (31, 32, 33, and 42 in column 5) show a higher reducing level of 53.5% corresponding closely to what has been found as characteristic for Strep. faecalis species.

Although there have been a few strains, which by expanding the experimental limits may be classified as borderline cases, the majority of strains deviated decidedly from either of the reference groups tested.

Discussion

There are several points in this particular observation which require comment. First, it is apparent that the Enterococci found in blood do not necessarily belong to any one specific group. Yet, it is surprising to find that in spite of uniform biological and serological behavior the majority of strains deviate from the standard strains in the tetra-

zolium reduction test. This could mean either that these freshly isolated strains differ from old culture strains, or that they are genuinely distinct in this particular property. Be that as it may, the method permits a relatively simple way of characterization of unknown Enterococcus strains. This is in general agreement with the findings of Barnes, but she studied strains from different sources.

Summary

The tetrazolium reduction test was applied to Enterococci (Lancefield group D) derived from rheumatic fever patients and compared with standard strains belonging to the four Sherman groups. Twenty-eight strains did not correspond with either of these groups but showed a distinct behavior. Only 4 strains showed reducing capacities analogous to Strep. faecalis.

REFERENCES

1. Laxminarayana, H. and Iya, K. U.: Indian J. Dairy Sci., 1953, vol. 6, *75*.
2. Barnes, E. M.: J. Gen. Microbiol., 1956, vol. 14, *57-68*.
3. Dias, V. M. and Niber da Paz M. da Silva: Ztb. f. Bakt. I, O., 1958, vol. 171, *317*.
4. Scheff, G. J., Marienfeld, C., and Hackett, E. J.: Inf. Dis., 1958, vol. 103, *45*.
5. Sherman, J. M.: Bact. Rev., 1937, vol. 1, *1-37*.

« ABSTRACTS »

COLOMBO, J-P., WEBER, J. W., GUIDOTTI, G., KANAMEISHI, D., and FOA, P. P. (Dept. of Physiology and Pharmacology, The Chicago Medical School): Liver Phosphorylase in Normal and Adrenalectomized Dogs Treated with Serotonin. Endrocrinology 67(5): 693-697, 1960.

The hyperglycemic action of serotonin was investigated in 6 normal and 10 adrenalectomized dogs. In normal dogs, intravenous injections of serotonin were followed promptly by hyperglycemia, decreased liver glycogen and increased liver phosphorylase activity. In control adrenalectomized dogs treated with saline, a proqressive decrease in blood glucose and liver glycogen was observed. Serotonin injections did not prevent these phenomena and caused only a delayed increase in phosphorylase activity.

These results are consistent with the hypothesis that the increase of liver phosphorylase activity, glycogenolysis, and blood glucose concentration observed in normal dogs are not due to a direct stimulation of liver phosphorylase by serotonin, but are secondary to a release of epinephrine from the adrenal glands.

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FOA, P. P., and GALANSINO, G. (Dept. of Physiology and Pharmacology, The Chicago Medical School): Glucagon. Clin. Endocrinol., 1:269-277, 1960.

A review with 80 references.

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COLOMBO, J-P., WEBER, J. W., KANAMEISHI, D., and FOA, P. P. (Dept. of Physiology and Pharmacology, The Chicago Medical School): Site of Origin of a Hyperglycemic Substance Liberated into the Pancreato-Duodenal Blood by Deserpidine Injections. Endocrinology 67(2): 248-251, 1960.

The injection of deserpidine into the pancreatoduodenal artery of normal dogs is followed by a prompt rise in blood sugar concentration. This hyperglycemia is not observed in previously depancreatized dogs. The results suggest that deserpidine causes the release of a hyperglycemic substance originating in the pancreas. The nature of this substance is unknown, although it appears to be different from the hyperglycemic material released by the injection of growth hormone.

GALANSINO, G., D'AMICO, G., KANAMEISHI, D., BERLINGER, F. G., and FOA, P. P. (Dept. of Physiology and Pharmacology, The Chicago Medical School): Hyperglycemic Substances Originating in the Pancreatoduodenal Area. Am. J. Physiol. 198(5): 1059-1062, 1960.

Glucagon, serotonin, deserpidine and the diethanolamine salt of p-tolymethyl carbinol camphoric acid ester (DTCE) were injected into the pancreatic, mesenteric or femoral artery, or into the portal or femoral vein of anesthetized normal dogs pretreated with dihydroergotamine (DHE) or with SA-97, a serotonin-blocking agent. Intraportal glucagon causes a fleeting, but marked and significant, elevation of plasma potassium and a marked and significant hyperglycemia. DHE blocks glucagon hyperkalemia, but not glucagon hyperglycemia.

These effects are similar to the effects of glucagon injected into a peripheral vein and suggest that glucagon hyperkalemia may be mediated by a sympathomimetic amine. Serotonin causes a marked hyperglycemia which is not seen in animals pretreated with DHE or SA-97. Deserpidine causes an immediate and marked hyperglycemia when injected into the pancreatic artery, and a mild and delayed hyperglycemia if injected elsewhere. DTCE also causes hyperglycemia only when injected into the pancreatic artery. DHE blocks the effect of serotonin and of DTCE, but not that of deserpidine.

It is suggested that more than one type of hyperglycemic substance may be produced in the area supplied by the pancreatic artery: one type which, like serotonin or the catecholamines, is blocked by DHE, and another type which, like glucagon, is unaffected by it.

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GUIDOTTI, G., COLOMBO, J-P., and FOA, P. P. Dept. of Physiology and Pharmacology, The Chicago Medical School): Enzymatic Determination of Glucose. Stabilization of Color Developed by Oxidation of o-Dianisidine. Anal. Chem., 33: 151, 1961.

A sensitive method for the determination of glucose is described.